central estimates and 95% confidence intervals (in parentheses) of the excess relative risk (ERR) of thyroid cancer per Gy in children have been reported:

- 4.7 (1.7, 11) – atomic-bomb survivors less than 15 years old at time of exposure, with a mean thyroid dose of gamma rays of 0.27 Gy (Ron et al., 1995);
- 9.5 (4.1, 19) – atomic-bomb survivors less than 10 years old at time of exposure, with the same mean thyroid dose (Thompson et al., 1994).

These estimates suggest that the risk to children of age less than 10 years at time of exposure is greater than the risk to children of age 10-15 years. For children exposed to *X* rays, the following estimates of the ERR of thyroid cancer per Gy have been reported (Ron et al., 1995):

- 9.1 (3.6, 29) – newborn children in Rochester, New York, treated for an enlarged thymus gland at ages less than 1 year, with a mean thyroid dose of *X* rays of 1.4 Gy;
- 33 (14, 57) – Israeli children treated for ringworm of the scalp at a mean age of 7 years, with a mean thyroid dose of *X* rays of 0.09 Gy;
- 2.5 (0.6, 26) – children in Chicago, Illinois, treated for enlarged tonsils and adenoids at ages 0-15 years (mean age of 4 years), with a mean thyroid dose of *X* rays of 0.59 Gy;
- 7.7 (2.1, 29) – pooled analysis of data on childhood exposures at ages 0-15 years, including data on the atomic-bomb survivors (result is dominated by data on childhood exposures to *X* rays at ages less than 10 years).

An RBE for *X* rays can be estimated from these results by assuming that the probability distribution of the estimated risk in each study is lognormal and calculating ratios of the probability distributions for *X* rays to a distribution for gamma rays. Since the average ages of children exposed to *X* rays were 7 years or less, we use the estimated risk in the atomic-bomb survivors of age less than 10 years at time of exposure to estimate RBEs. The 95% confidence intervals of RBEs obtained from the three studies of children exposed to *X* rays are (0.3, 4.2), (1.1, 9.2), and (0.06, 3.5), and the 95% confidence interval obtained using the results of the pooled analysis is (0.2, 4.0). If we assume that the biological effectiveness of *X* rays in humans should not be less than that of high-energy gamma rays, based on the effective quality factor shown in Fig. 1 (ICRU, 1986), these confidence intervals indicate that the RBE of *X* rays in inducing thyroid cancer in children most likely is in the range of about 1-4. However, the estimated risks in the different populations neither support nor refute an assumption that *X* rays are more effective than high-energy gamma rays in inducing thyroid cancer in children.

Additional information on the RBE for *X* rays and thyroid cancer can be obtained from a study of prepubescent rats exposed to *X* rays and beta particles emitted in ^{131}I decay (Lee et al., 1982). As discussed in a later section, the biological effectiveness of ^{131}I beta particles, which have an average energy of 182 keV (Kocher, 1981), should be similar to that of high-energy gamma rays. The following central estimates and 95% confidence intervals (in parentheses) of ratios of thyroid tumor incidence from exposure to *X* rays to tumor incidence from exposure to ^{131}I beta particles were obtained: 1.1 (0.32, 3.7) at a mean thyroid dose of 0.8 Gy, 1.2 (0.43, 3.2)

at 3.3 Gy, and 1.4 (0.24, 7.6) at 8.5 Gy. The average of the three confidence intervals is a distribution with a central estimate (50^{th} percentile) and 95% confidence interval of 1.4 (0.6, 3.6). Thus, although the uncertainties are large and an RBE for *X* rays as high as about 4 cannot be ruled out, the biological effectiveness of *X* rays and ^{131}I beta particles in inducing thyroid cancer in the study animals was about the same, on average.

Finally, we examined results obtained from epidemiological studies of cancers at other sites, including the colon, lung, skin, female breast, and bladder (UNSCEAR, 2000). The central estimate of the ERR per Gy in populations exposed to *X* rays often was comparable to or less than the central estimate in a similar age group in the atomic-bomb survivors, although some of the lower risks from *X* rays may be influenced by the much higher doses of *X* rays compared with the doses of gamma rays in the atomic-bomb survivors. In those few cases where a higher risk was observed in populations exposed to *X* rays, the difference was less than a factor of 2. In all cases, however, uncertainties in the risk estimates are sufficiently large that an RBE for *X* rays substantially greater than 1 cannot be ruled out. Thus, as in the case of thyroid cancer, the available data on other cancers in humans do not indicate whether *X* rays are biologically more effective than high-energy gamma rays or not.

The results of epidemiological studies described above lead to the following observations. First, there is no evident difference in the effectiveness of *X* rays in inducing thyroid cancers compared with cancers at other sites. Second, uncertainties in the results of epidemiological studies are sufficiently large that an upper confidence limit of REF_L as high as 5.0, as we have assumed based on radiobiological data, cannot be ruled out. Third, although uncertainties in the results of epidemiological studies are large, in no cases is a central estimate of an RBE for *X* rays as high as 4 obtained. Based on considerations of statistical uncertainties alone, an occasional high estimate of RBE would be expected. Finally, the epidemiological data do not rule out an assumption that the biological effectiveness *X* rays in inducing cancers in humans is the same as that of high-energy gamma rays.

Based on the evidence obtained from all the radiobiological and epidemiological studies discussed above and an assumption that the biological effectiveness of *X* rays should not be substantially less than that of high-energy gamma rays, we describe REF_L for orthovoltage *X* rays and other lower-energy photons by the following hydrid probability distribution:

[1] 75% weight to a lognormal distribution having a 95% confidence interval between 1.0 and 5.0, based on the results of radiobiological studies;

[2] 25% weight to the value 1.0, based on the lack of clear evidence of a difference between *X* rays and gamma rays in epidemiological data.

Thus, we use the results of epidemiological studies to modify the lognormal probability distribution that was based on the results of radiobiological studies by assigning a substantial weight to an assumption that *X* rays and other lower-energy photons have the same biological

effectiveness in humans as high-energy gamma rays. The resulting probability distribution of REF_L has a median of 1.9 and a 95% confidence interval between 1.0 and 4.7. The cumulative probability distribution of REF_L is shown in Fig. 18. The assumption that this distribution applies at photon energies of 30-250 keV is discussed below.

The weight to be given to an assumption that *X* rays and other lower-energy photons have the same biological effectiveness in humans as high-energy gamma rays clearly is a matter of subjective judgment. Our judgment is that the weight given to this assumption should be substantial but should not be much higher than 25% (e.g., 50% or higher). This judgment is based on two considerations. First, there are many and varied radiobiological studies which clearly indicate a greater biological effectiveness of *X* rays. Second, although the epidemiological data do not show clear evidence of a difference in biological effectiveness, the data are so uncertain that differences as large as a factor of 3 cannot be ruled out.

Energy Dependence of REF

Based on the energy dependence of the effective quality factor shown in Fig. 1 (ICRU, 1986), we assume that the probability distribution of REF_L for orthovoltage *X* rays and other lower-energy photons described above applies at energies of 30-250 keV; the effective quality factor is essentially independent of energy over much of this range. We also note that the effective quality factor at these energies is approximately twice the value at the energies of ^{60}Co gamma rays; by our reading of the curve in Fig. 1, the difference is a factor of 2.3. The ICRU's calculation thus supports our assumed distribution of REF_L.

An assumption that the distribution of REF_L applies at photon energies as low as 30 keV is supported by calculations of the biological effectiveness of 60- and 80-kVp *X* rays relative to gamma rays from the Hiroshima and Nagasaki atomic bombs for a number of specific endpoints, including chromosomal aberrations in human lymphocytes, induction of mutations in human fibroblasts, and oncongenic transformation in C3H10T½ mouse cells (Brenner, 1999). RBEs at low doses between 1.6 and 2.0 were calculated. The differences between these values and the value of 2.3 inferred from the calculation in Fig. 1 are due, in part, to differences in the assumed responses as a function of lineal energy and to an assumption that the average energies of gamma rays from the atomic bombs were somewhat less than the energies of ^{60}Co gamma rays.

The effective quality factor shown in Fig. 1 also indicates that the biological effectiveness of photons increases as the energy decreases below 30 keV. For example, based on the calculation in Fig. 1, Brenner and Amols (1989) estimated that 23 kVp *X* rays should be approximately 1.3 times more effective than 44-250 kVp *X* rays in inducing breast cancer. Thus, we assume that the probability distribution of REF_L for photons of energy 30-250 keV should be increased when the energy is less than 30 keV. We represent this increase by a factor which is described by a triangular probability distribution having a lower bound of 1.0, a mode of 1.3, and an upper bound of 1.6. The resulting probability distribution of REF_L at photon energies less than 30 keV is shown in Fig. 19.

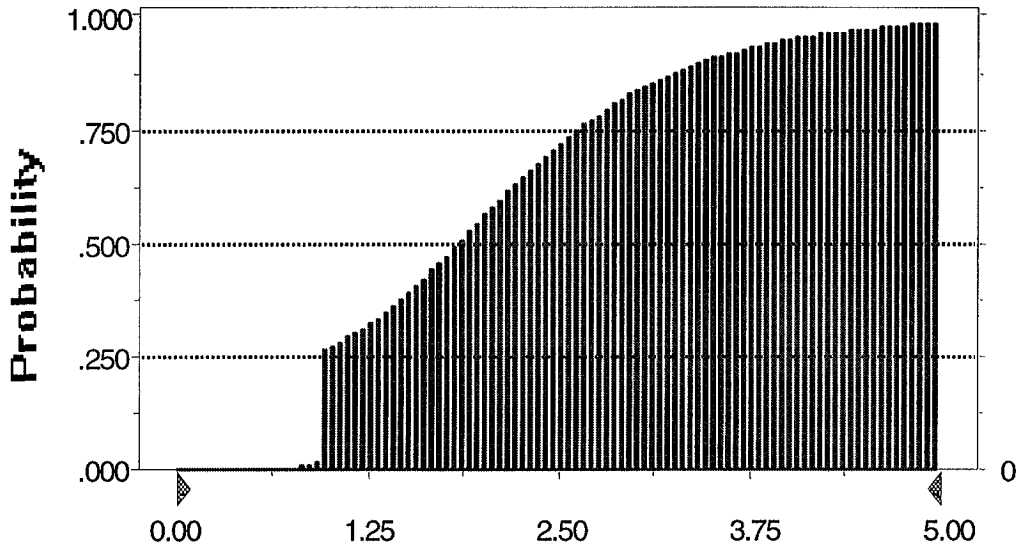

Fig. 18. Assumed probability distribution of REF_L for photons of energy 30-250 keV (25% weight to value 1.0; 75% weight to lognormal probability distribution having a 95% confidence interval between 1.0 and 5.0) displayed as a cumulative distribution; distribution applies to all cancers and at any dose and dose rate. Median of distribution is 1.9, and 95% confidence interval lies between 1.0 and 4.7; about 1.8% of values lie beyond 5.0.

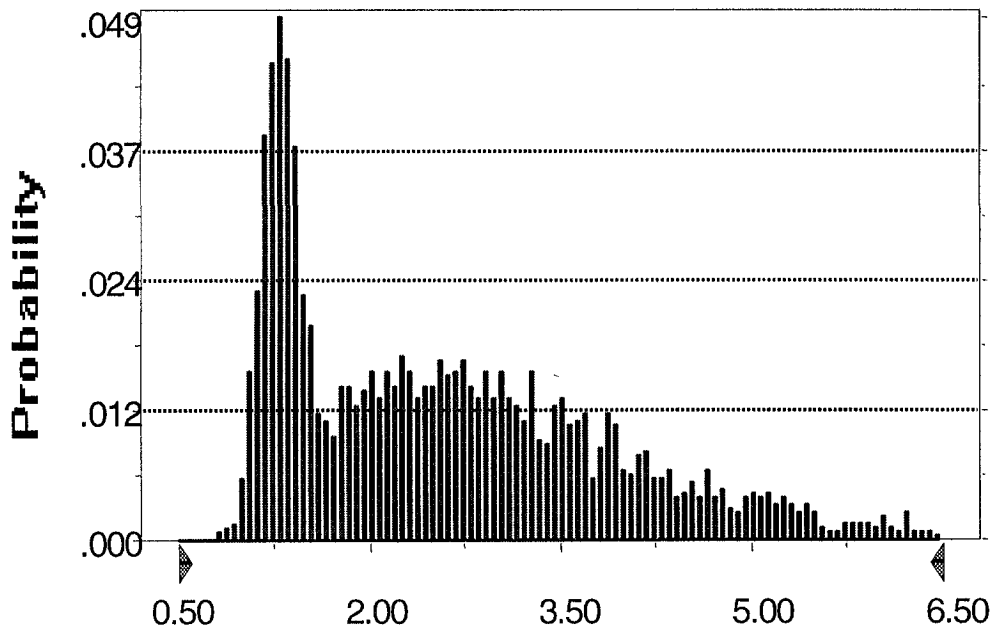

Fig. 19. Assumed probability distribution of REF_L for photons of energy less than 30 keV; distribution applies to all cancers and at any dose and dose rate. Median of distribution is 2.4 and 95% confidence interval lies between 1.1 and 6.1; about 1.8% of values lie beyond 6.5.

Summary

The biological effectiveness of lower-energy photons relative to high-energy gamma rays is assumed to be independent of dose and dose rate under similar conditions of exposure to the two radiations. Cancer risks in humans from exposure to photons (γ) are estimated using the following equations:

Solid tumors (any dose and dose rate) –

$$\Re_\gamma = \mathrm{REF}_{\gamma,\mathrm{L}} \times \mathrm{AF}_\gamma \times \frac{R_{\gamma,\mathrm{H}}}{\mathrm{DDREF}_\gamma} \times D_\gamma \qquad (11)$$

Leukemias (acute exposure) –

$$\Re_\gamma = a(\mathrm{REF}_{\gamma,\mathrm{L}} \times \mathrm{AF}_\gamma \times D_\gamma) + b(\mathrm{REF}_{\gamma,\mathrm{L}} \times \mathrm{AF}_\gamma \times D_\gamma)^2 \qquad (12)$$

Leukemias (chronic exposure) –

$$\Re_\gamma = a \times \mathrm{REF}_{\gamma,\mathrm{L}} \times D_\gamma \qquad (13)$$

where $\mathrm{REF}_{\gamma,\mathrm{L}}$ is the radiation effectiveness factor at low doses and low dose rates that applies at photon energies of 30-250 keV, AF_γ is an adjustment factor representing an increase in biological effectiveness at photon energies less than 30 keV, $R_{\gamma,\mathrm{H}}$ is the risk coefficient (ERR per Gy) at high acute doses of high-energy gamma rays for the solid tumor of concern, a and b are the coefficients of the linear and quadratic terms in the linear-quadratic dose-response relationship for leukemias at high acute doses of high-energy gamma rays, DDREF_γ is the dose and dose-rate effectiveness factor for gamma rays and other low-LET radiations, and D_γ is the absorbed dose of photons in the organ or tissue of concern. The value of REF_L for all photons of energy greater than 250 keV is assumed to be unity. The probability distribution of REF_L at a given photon energy is applied to all cancers.

A single probability distribution of DDREF_γ is applied in estimating risks of solid tumors under conditions of chronic exposure. Under conditions of acute exposure, DDREF_γ depends on the magnitude of the dose. At acute doses greater than 0.2 Gy, DDREF_γ is assumed to be unity. At acute doses less than 0.2 Gy, a DDREF_γ that can exceed unity is applied, and the distribution of possible values approaches the probability distribution of DDREF_γ under conditions of chronic exposure as the dose approaches zero (Land et al., 2002).

The assumed probability distributions of REF_L for photons of different energies are summarized in Table 12.

Table 12. Summary of probability distributions of radiation effectiveness factors for photons to be used in estimating cancer risks and probability of causation in accordance with eq. (11), (12), or (13)[a]

Photon energy	Probability distribution of radiation effectiveness factor (REF_L)
> 250 keV	Single-valued at 1.0 (higher-energy photons are assumed reference radiation)
30-250 keV	Hybrid distribution with – 75% weight to lognormal distribution having a 95% confidence interval between 1.0 and 5.0; 25% weight to value 1.0;
< 30 keV	Product of two distributions – (1) hybrid distribution for photons of energy 30-250 keV; and (2) triangular distribution with minimum of 1.0, mode of 1.3, and maximum of 1.6

[a]Probability distributions apply to all cancers and at any dose and dose rate.

ELECTRONS

With the exception of low-energy electrons emitted in beta decay of ^3H, there have been few studies of the biological effectiveness of electrons relative to gamma rays or *X* rays. In this section, we develop a probability distribution of an REF for beta particles emitted in ^3H decay; these electrons have an average energy of 5.7 keV and a maximum energy of 18.6 keV (Kocher, 1981). We then consider the biological effectiveness of other electrons, including low-energy Auger electrons.

REF for Tritium Beta Particles

Many studies have shown that beta particles emitted in decay of ^3H are biologically more effective than gamma rays in inducing stochastic effects (NCRP, 1990; Straume and Carsten, 1993). Estimates of RBE obtained from studies reviewed by Straume and Carsten (1993), including studies in which the reference radiation was *X* rays, are summarized in Tables 13-16.

For purposes of developing an REF for ^3H beta particles that is consistent with the REFs for the other radiation types, the relevant studies are those in which the reference radiation was gamma rays. In most studies using gamma rays, the reference radiation was delivered chronically to match the conditions of exposure to ^3H beta particles. Thus, cancer risks in humans from

exposure to ^3H beta particles are estimated using the models represented in eqs. (2) and (5), which apply at low doses and low dose rates. If we assume that DDREF for ^3H beta particles in the various studies is about the same as DDREF for the reference radiation, RBEs obtained under conditions of chronic exposure in Tables 13-16 provide estimates of RBE_M.

Based on the data under conditions of chronic or sub-acute exposure to gamma rays in Tables 13-16, but excluding the data for tritiated thymidine, central estimates of RBE_M are in the range of about 1.5-3. When uncertainties in these estimates are considered, the range presumably is about 1.2-4. The data on RBE for tritiated thymidine are not included based on the consideration that this compound has a different distribution within the body than other chemical forms of ^3H that would be encountered in the workplace or the environment.

Estimates of RBE_M under conditions of chronic exposure to *X* rays also are relevant. In three of the five such determinations, the central estimate of RBE_M is about 2-3. If we assume a nominal biological effectiveness of *X* rays relative to gamma rays of 2, based on the probability distribution of REF_L for *X* rays developed in the previous section, these data indicate that RBE_M for ^3H beta particles relative to gamma rays is as high as about 4-6.

Table 13. Estimates of RBE of tritium beta particles for carcinogenesis endpoints[a]

Effect	Radiation and conditions	RBE	Reference
Mammary tumors in S-D rats	HTO and chronic *X* rays	1.2 ± 0.3	Gragtmans et al. (1984)
Leukemia in CBA/H mice	HTO and chronic *X* rays	1.2 ± 0.3	Myers and Johnson (1991)
Tumors in C57B1/6N × C3H/He mice	HTO and acute gamma rays	~1[b]	Yokoro et al. (1989)
Transformation in hamster cells *in vitro*	HTO and acute *X* rays	~1	Suzuki et al. (1989)
Transformation in mouse cells *in vitro*	HTO and acute *X* rays	1-2	Little (1986)
Transformation in 10T½ cells	HTO and subacute gamma rays	1.4-1.8	Yamaguchi et al. (1985)

[a]See Table 1 of Straume and Carsten (1993).
[b]Authors did not provide estimate of RBE but state that biological effectiveness of HTO and gamma rays was not very different.

Table 14. Estimates of RBE of tritium beta particles for genetic endpoints[a]

Effect	Radiation and conditions	RBE	Reference
6-Thioguanine resistance in mouse cells *in vitro*	HTO and chronic gamma rays	2.9	Ueno et al. (1989)
6-Thioguanine resistance in mouse cells *in vitro*	^{3}H-amino acid and chronic gamma rays	2.6	Ueno et al. (1989)
6-Thioguanine resistance in mouse cells *in vitro*	Tritiated thymidine (^{3}H-Tdr)[b] and chronic gamma rays	5.9	Ueno et al. (1989)
6-Thioguanine resistance in mouse cells *in vitro*	HTO and gamma rays at 10^{-5} mutant frequency		Nakamura et al. (1985)
	acute	1.5	
	chronic	2.4	
Chromosome aberrations in human sperm *in vitro*	HTO and chronic *X* rays	3	Kamiguchi et al. (1990)
Chromosome aberrations in fish lymphocytes *in vitro*	HTO and chronic gamma rays	1.9	Suyama and Etoh (1985)
Chromosome aberrations in mouse zygotes	HTO and chronic gamma rays	1.8	Matsuda et al. (1985)
Chromosome aberrations in CBA/H mice			Chopra and Heddle (1988)
lymphocytes	HTO and *X* rays	1.1	
spermatogonia	HTO and *X* rays	1.2	
Micronuclei in mammalian cells	HTO and chronic gamma rays	2.0 2.7	Ueno et al. (1982) Kashima et al. (1985)
Mutations in *Drosophila* spermatozoa	HTO and gamma rays	2.7	Byrne and Lee (1989)
Mutations in mice *in vivo*	HTO and chronic gamma rays	2.7	Nomura and Yamamoto (1989)

Table is continued on following page.

Table 14. Estimates of RBE of tritium beta particles for genetic endpoints (continued)[a]

Effect	Radiation and conditions	RBE	Reference
Dominant lethals in male mice	HTO and chronic gamma rays	2.5	Searle (1984)
		1-2	Carsten and Commerford (1976)
Dominant lethals in female mice	HTO and chronic gamma rays	2.5	Xiang-yan et al. (1986)
Specific locus mutations in male mice	HTO and chronic gamma rays	2.0	UNSCEAR (1982)

[a]See Table 2 of Straume and Carsten (1993).
[b]Use of methyl-^{3}H-Tdr resulted in identical RBEs.

Table 15. Estimates of RBE of tritium beta particles for chromosome aberrations in human lymphocytes[a]

Radiation and conditions	RBE	Reference
HTO and acute *X* rays	1.9 ± 0.7	Bocian et al. (1977), as refit by Prosser et al. (1983)
HTO and subacute gamma rays	1.49 ± 0.21	Morimoto et al. (1989)
HTO and acute *X* rays	1.13 ± 0.18	Prosser et al. (1983)
HTO and acute gamma rays	3.4 ± 0.6	Prosser et al. (1983) and Lloyd et al. (1975)
HTO and subacute *X* rays	2.6	Vulpis (1984)
HTO and low dose *X* rays	2.0	Estimated from Prosser et al. (1983) and Lloyd et al. (1988)

[a]See Table 3 of Straume and Carsten (1993).

Table 16. Estimates of RBE of tritium beta particles for developmental and related effects[a]

Effect	Radiation and conditions	RBE	Reference
Mouse embryo, two-cell to blastocyte *in vitro*	HTO and chronic gamma rays	1.7	Yamada et al. (1982)
Teratogenic effects in rat embryos	HTO and chronic gamma rays	2.6	Satow et al. (1989)
Cell killing *in vitro*	HTO and chronic gamma rays	1.3	Ueno et al. (1989)
	^{3}H-amino acids and chronic gamma rays	1.7	
	Tritiated thymidine (^{3}H-Tdr)[b] and chronic gamma rays	3.5	

[a]See Table 4 of Straume and Carsten (1993).
[b]Use of methyl-^{3}H-Tdr resulted in an identical RBE.

Taking into account the data on RBE relative to gamma rays and *X* rays, we describe the REF for ^{3}H beta particles at low doses and low dose rates, REF_L, by a lognormal probability distribution having a 95% confidence interval between 1.2 and 5.0. This distribution has a geometric mean (median) and geometric standard deviation of 2.4 and 1.4, respectively. Based on discussions presented below and except as noted, the probability distribution of REF_L for ^{3}H beta particles is assumed to apply to any electrons of energy less than 15 keV.

In a previous analysis by *SENES* Oak Ridge (Thomas and Hoffman, 2000), the biological effectiveness of ^{3}H beta particles was described by a triangular probability distribution having a lower bound of 1.0, a mode of 2.0, and an upper bound of 5.0. The lognormal probability distribution described above is similar to the previous assumption. However, the data summarized in Tables 13-16 indicate that an REF_L greater than 5 cannot be ruled out. The upper tail of the lognormal probability distribution represents an assumption that REF_L could be 5 or greater, and that a reasonable upper bound cannot be determined with certainty. The small probability assigned to an REF_L of 4 or greater (about 10%) also is intended to take into account that RBEs for organically-bound tritium appear to be 2-3 times higher than RBEs for HTO or ^{3}H incorporated into amino acids (see Tables 14 and 16). This is a potentially important consideration when some HTO taken into the body becomes organically-bound before it is excreted (Straume and Carsten, 1993).

In a recent analysis by Harrison et al. (2002) of the NRPB, the biological effectiveness of ^{3}H beta particles relative to high-energy gamma rays was described by a uniform probability distribution between 1.0 and 2.5; this distribution has a median of 1.75. The analysis by Harrison et al. differs from the analysis in this report mainly in two respects. First, these investigators evaluated RBEs for ^{3}H beta particles relative to high-energy gamma rays and RBEs relative to lower-energy *X* rays together without taking into account that *X* rays probably have a greater biological effectiveness than gamma rays. As discussed above, we have assumed that RBEs for ^{3}H beta particles relative to *X* rays should be increased by a factor representing the biological effectiveness of *X* rays to provide a proper comparison with RBEs relative to gamma rays. This adjustment by a factor of about two results in a median and upper confidence limit in our probability distribution of REF_L that are substantially higher than assumed by Harrison et al. Second, the uniform probability distribution developed by Harrison et al. incorporates an assumption that the biological effectiveness of ^{3}H beta particles relative to gamma rays could not exceed 2.5. In contrast, we believe that the data on RBE are consistent with values as high as about 5, and that an upper bound near 5 cannot be established with certainty. These assumptions are incorporated in the lognormal probability distribution of REF_L developed in this report.

The assumed lognormal probability distribution of REF_L for ^{3}H beta particles having a 95% confidence interval between 1.2 and 5.0 is nearly the same as the probability distribution of REF_L for photons of energy less than 30 keV discussed in the previous section. This consistency is expected when, as discussed below, the energies of electrons that deliver an absorbed dose are similar in the two cases.

Consideration of Energy Dependence of REF

Since the energies of ^{3}H beta particles are very low, we also considered whether other electrons, especially those of higher energy, should have an REF greater than unity. In radiation protection, all such electrons generally are assumed to have the same biological effectiveness as high-energy gamma rays (see Table 1). A study of the biological effectiveness of *X* rays and beta particles from ^{131}I decay in inducing thyroid cancer in rats by Lee et al. (1982) discussed previously is the only study we are aware of that was designed to investigate the biological effectiveness of higher-energy electrons. In the absence of extensive radiobiological data, we address this question using the following arguments.

In the previous section, data on the biological effectiveness of *X* rays and a calculation of the energy dependence of the effective quality factor for photons shown in Fig. 1 were used to develop a probability distribution of REF_L with a median greater than 1.0 for photons of energy as high as 250 keV. Since the absorbed dose from irradiation by photons is due almost entirely to energetic secondary electrons produced by interactions of the incident photons in tissue, information on the biological effectiveness of photons can be used to infer the biological effectiveness of electrons. That is, an REF for photons of a given energy essentially describes the biological effectiveness of the secondary electrons produced by the first interactions of the photons in tissue.

The energies of secondary electrons produced by interactions of photons in tissue generally decrease with decreasing photon energy. Therefore, electrons produced by interactions of 250-keV photons in tissue are at the highest energies for which the biological effectiveness should be the same as that of lower-energy photons. In tissue, which has an average atomic number of 7 (Shleien et al., 1998), Compton scattering is the dominant interaction at a photon energy of 250 keV [see Fig. A.1 of NCRP (1991) and Figs. 5.1 and 5.2 of Shleien et al. (1998)]. At this energy, the continuous spectrum of secondary electrons produced by Compton scattering has a maximum energy of 124 keV and an average energy of 60 keV (Turner, 1995). In contrast, the energy of secondary electrons produced by the photoelectric effect in tissue at this energy is nearly 250 keV, since the binding energies of electrons in atoms of the elements comprising tissue are about 3 keV or less (Shleien et al., 1998). At 250 keV, however, photoelectrons are produced in only about 0.1% of all interactions [see Fig. 5.2 of Shleien et al. (1988)] and, thus, have little effect on the average energy of secondary electrons.

As the incident photon energy decreases below 250 keV, the photoelectric effect increases in importance relative to Compton scattering and becomes the dominant interaction in tissue at energies less than about 30 keV (Schleien et al., 1998; NCRP, 1991). At this photon energy, the average and maximum energies of secondary electrons produced in Compton scattering are about 1.5 and 3 keV, respectively, and the average energy of photoelectrons is nearly 30 keV. Thus, the average energy of secondary electrons produced by 30-keV photons is about 15 keV. This result is of interest because we have assumed, based on the calculated energy dependence of the effective quality factor shown in Fig. 1, that the biological effectiveness of photons of energy less than 30 keV is higher than at 30-250 keV. Thus, the same increase in biological effectiveness should apply to electrons of energy less than about 15 keV. As the photon energy decreases below 30 keV, the average energy of secondary electrons produced in tissue approaches the incident photon energy, due to the increasing importance of the photoelectric effect and the low binding energies of atomic electrons. At a photon energy of 20 keV, for example, the energies of secondary electrons are little different from the incident photon energy.

Three conclusions can be drawn from the analysis described above. First, at energies greater than about 60 keV, the biological effectiveness of electrons should be essentially the same as that of the reference high-energy gamma rays (i.e., unity). Second, at energies in the range of about 15-60 keV, the biological effectiveness of electrons should be the same as that of photons of energy 30-250 keV. Third, at energies less than about 15 keV, but possibly excluding Auger electrons as discussed below, the biological effectiveness of electrons should be the same as that of photons of energy less than 30 keV.

We use the conclusions about the biological effectiveness of electrons of various energies in the following way. First, the assumed probability distribution of REF_L for beta particles from ^3H decay is applied to any electrons of energy less than 15 keV. In general, this REF_L should be applied to electrons from beta decay when the average energy of the spectrum of beta particles is less than 15 keV. Use of the average energy of beta particles is reasonable when the argument to assume an REF_L greater than 1.0 at energies less than 15 keV was based in part on the average

energy of the spectrum of secondary electrons in Compton scattering. The assumed REF_L also should be applied to discrete internal conversion electrons of energy less than 15 keV emitted in radioactive decay.[21] In these cases, however, an increased biological effectiveness needs to be taken into account only when the average energy of low-energy internal conversion electrons per decay of a radionuclide is significant compared with the average energies per decay of other radiations that have a short range in tissue, including internal conversion electrons of energy greater than 15 keV, beta particles, and alpha particles. Application of the assumed REF_L to Auger electrons is discussed below.

Second, we have not adopted an increased biological effectiveness of 15-60 keV electrons relative to high-energy photons, even though the calculations described above indicate that REF_L for electrons in this energy range should be the same as that for 30-250 keV photons. This decision was based on two considerations noted previously. First, we are not aware of any data on RBE for 15-60 keV electrons. Second, the assumed REF_L for 30-250 keV photons is based on studies in which the average energies of *X* rays was about 50-65 keV and relatively few *X* rays had energies above 100 keV, and the assumption that RBEs for such *X* rays apply at photon energies up to 250 keV was based on a calculation of the energy dependence of the effective quality factor shown in Fig. 1. Thus, adoption of an REF_L greater than unity for 15-60 keV electrons would be based on two assumptions for which there is no experimental evidence. In general, we have assumed that probability distributions of REFs should be developed only when there is some basis in radiobiological data.

Biological Effectiveness of Auger Electrons

Radionuclides that emit Auger electrons[22] require special consideration, due to the very low energies of these radiations (often a few keV or less) and their short range in matter (less than 0.1 μm). The ICRP (1991) and the NCRP (1993) recommend that Auger electrons emitted by radionuclides that are incorporated into DNA should not be assigned a radiation weighting factor of 1 (see Table 1), since it is unreasonable to average the absorbed dose over the whole mass of DNA. Techniques of microdosimetry are considered more appropriate in such cases.

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[21]Internal conversion is the process by which the energy difference between an initial and final state in an atomic nucleus is transferred directly to a bound atomic electron, which is then ejected from the atom (NCRP, 1985). Emission of internal conversion electrons competes with emission of gamma rays, and it increases in importance as the atomic number increases and the transition energy decreases.

[22]The emission of Auger electrons competes with the emission of *X* rays as a means of carrying off the energy released when a vacancy in an inner atomic shell of electrons, created by an electron capture or internal conversion electron event, is filled by an electron from an outer shell (NCRP, 1985). In the Auger process, the filling of a vacant inner shell is accompanied by the simultaneous ejection of one or more electrons from an outer shell. Auger electrons are important compared with other radiations having a short range in matter (beta particles, internal conversion electrons, and alpha particles) mainly when a radionuclide decays by electron capture or an isomeric transition (Kocher, 1981).

Limited data on the biological effectiveness of low-energy Auger electrons are summarized by the ICRP (1991). When an Auger-emitting radionuclide penetrates a cell but is not incorporated into DNA, estimated RBEs for a number of endpoints, including cell killing, are in the range of 1.5-8. These RBEs are similar to estimates for low-energy beta particles emitted in ^{3}H decay discussed previously. When Auger emitters, such as ^{125}I, are incorporated into DNA, RBEs in the range of 20-40 have been estimated for such endpoints as cell transformation. These high RBEs are supported by calculated patterns of energy deposition.

When information on whether an Auger-emitting radionuclide is incorporated into DNA of an exposed individual is lacking, we believe that Auger electrons should be treated in the same way as other low-energy electrons. Thus, for example, when the energy of Auger electrons is less than 15 keV, the probability distribution of REF_L that applies to low-energy beta particles emitted in decay of ^{3}H should be used. When Auger electrons are important compared with other low-energy electrons, their energies are nearly always less than 15 keV (Kocher, 1981).

When an Auger-emitting radionuclide is known to be incorporated into DNA, however, we do not believe that a credible probability distribution of REF_L can be developed based on available information. Although REF_L in such cases should be substantially higher than the REF_L that applies to ^{3}H beta particles, there are potentially important uncertainties including, for example, the fraction of the activity that is incorporated into DNA, the dependence of RBE on the energy of Auger electrons, and the dependence of RBE on dose when cell killing could occur. Thus, we support the recommendation of the ICRP (1991) and the NCRP (1993) that the biological effectiveness of Auger emitters that are incorporated into DNA should be handled as special cases using techniques of microdosimetry.

Summary

The biological effectiveness of low-energy electrons relative to high-energy gamma rays is assumed to be independent of dose and dose rate under similar conditions of exposure to the two radiations. Cancer risks in humans from exposure to electrons (e) are estimated using the following equations:

Solid tumors (any dose and dose rate) –

$$\Re_e = \mathrm{REF}_{e,\mathrm{L}} \times \frac{R_{\gamma,\mathrm{H}}}{\mathrm{DDREF}_{\gamma}} \times D_e \qquad (14)$$

Leukemias (acute exposure) –

$$\Re_e = a(\mathrm{REF}_{e,\mathrm{L}} \times D_e) + b(\mathrm{REF}_{e,\mathrm{L}} \times D_e)^2 \qquad (15)$$

Leukemias (chronic exposure) –

$$\Re_e = a \times \mathrm{REF}_{e,\mathrm{L}} \times D_e \qquad (16)$$

where $\mathrm{REF}_{e,\mathrm{L}}$ is the radiation effectiveness factor at low doses and low dose rates, $R_{\gamma,\mathrm{H}}$ is the risk coefficient (ERR per Gy) at high acute doses of high-energy gamma rays for the solid tumor of concern, a and b are the coefficients of the linear and quadratic terms in the linear-quadratic dose-response relationship for leukemias at high acute doses of high-energy gamma rays, DDREF_γ is the dose and dose-rate effectiveness factor for gamma rays and other low-LET radiations, and D_e is the absorbed dose of electrons in the organ or tissue of concern. A probability distribution of REF_L based on RBEs for beta particles emitted in decay of ^{3}H is applied at all electron energies less than 15 keV (i.e., when the average energy of beta particles or the energy of discrete electrons is less than 15 keV), except the distribution does not apply when an Auger-emitting radionuclide is known to be incorporated into DNA. The probability distribution of REF_L for low-energy electrons is applied to all cancers. The value of REF_L for all electrons of energy greater than 15 keV is assumed to be unity.

As described previously in the summary for photons, a single probability distribution of DDREF_γ is applied in estimating risks of solid tumors under conditions of chronic exposure to electrons, and DDREF_γ under conditions of acute exposure depends on the magnitude of the dose (Land et al., 2002). Acute exposure to beta particles and other electrons emitted by radionuclides is not expected to be of concern. For example, given the residence half-time of tritiated water in soft tissues of about 10 days (ICRP, 1979) and the longer half-life of ^{3}H (12.3 years), exposures to beta particles emitted by ^{3}H generally should be chronic. Acute exposure to electrons presumably is a concern only in situations involving unusual external exposures.

The assumed REF_L for low-energy electrons would be important in calculating cancer risks and probability of causation whenever intakes of radionuclides that emit low-energy beta particles, internal conversion electrons, or Auger electrons contribute significantly to estimated doses to an organ or tissue of concern. Examples of potentially important radionuclides include, in addition to ^{3}H, the beta-emitting radionuclides ^{106}Ru and ^{107}Pd and the Auger-emitting radionuclides ^{51}Cr, ^{55}Fe, ^{57}Co, ^{58}Co, ^{65}Zn, and ^{125}I (Kocher, 1981).

Based on considerations of the energies of secondary electrons produced by interactions of photons in tissue and the assumed REF_L for photons of energy 30-250 keV, REF_L also should be greater than unity at electron energies of 15-60 keV. However, such an REF_L is not adopted in this work, due to the lack of supporting radiobiological data.

The assumed probability distributions of REF_L for electrons of different energies are summarized in Table 17.